Musser Compliments of the Authors.

NOTES OF A CASE

OF

INFECTIOUS, SO-CALLED ULCERATIVE ENDOCARDITIS, AND OF A CASE OF ACUTE PERICARDITIS.

BY

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1110 Spring Garden St.

EXTRACTED FROM THE

TRANSACTIONS OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA,

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THE following case of endocarditis of the infectious or ulcerative variety is presented for your consideration, to-night, on account of its comparative rarity, and the extreme interest that is aroused by some striking features in its cause, its course, and its anatomical characters. The patient was under the care of Dr. Ludlow in the Presbyterian Hospital, and our obligations are due him for the privilege of this presentation. The following is an abstract of the ward notes of Dr. Hamaker.

CASE.—Infectious endocarditis secondary to acute rheumatism; intermitting fever; sweats; diarrhœa; pneumonia. Death. Proliferative endocarditis; micrococci; renal emboli.

W. J., white, æt. 30, baker, was admitted to the medical wards of the Presbyterian Hospital, October 3, 1884, suffering from rheuma-

tism, localized in the left knee and the right shoulder and elbow. He stated that he had been feeling badly for some time, losing flesh and strength. Previous to the present illness, he had had smallpox, syphilis, and gonorrhœa.

In addition to the joint pain, it was found he had a temperature of 104°, oppression behind the sternum, with pain aggravated by a slight cough. His face was pock-marked; he had the appearance of a drinking man, and was extremely anæmic. No cardiac murmur detected.

On October 10th, one week after admission, it was noted the cough and substernal distress had disappeared, the temperature had fallen to the normal, the joint pains were much relieved. The joints had not been swollen or red, but were painful. It was, at this time, thought worthy to note the occurrence of profuse sweats, both day and night.

Oct. 21. Fever and joint trouble recurred; sweating continues; occasional diarrhœa, soft systolic murmur noted at the apex.

25*th.* Nose bled profusely, requiring plugging of the right nostril.

Nov. 1. Pneumonia of lower lobe of right lung. Mitral and aortic murmur heard. No joint pains.

19*th.* Resolution of the pneumonic consolidation; return of joint symptoms.

Dec. 9. Joint symptoms increased in severity and became general. The fever has continued intermitting in type (*see* Chart), the sweating more marked, the pulse and respirations extremely rapid. Past two days, congestion of both lungs. Death at 6 P.M., to-day (9th), or about ten weeks after admission.

Autopsy, twenty-two hours after death.—Head and joints not examined. Bloodless appearance of face and extremities; emaciation of slight degree; marked rigor mortis; large ecchymoses on back of trunk and legs; pin-head size purpuric spots over trunk and extremities, anterior.

Thorax.—Lungs œdematous, and at bases, posterior, the seat of hypostatic congestion; pieces float on water; a few hemorrhagic infarcts are seen in the middle lobes.

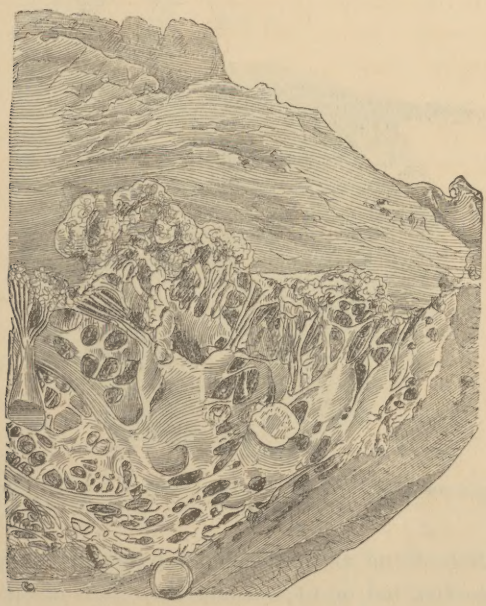
Microscopical Examination.—The results of an inflammatory action are everywhere present. The interalveolar connective tissue,

especially in the immediate vicinity of the bloodvessels, is greatly increased, and contains a notable number of new cells, as well as quantities of dark pigment. The alveoli are generally distended, being partially filled with shed proliferated lining epithelium, while their walls are thickened.

The pleuræ were healthy in appearance. The pericardium was normal; its sac contained a small amount of serum.

Heart.—Slightly hypertrophied. Its exterior appeared normal; on section the walls were normal, and but slightly increased in thick-

FIG. 1.



Mitral valves, showing inflammatory outgrowths.

ness. In both ventricles, ante-mortem clots were seen extending far into their respective vessels. On removal of the clot from the left ventricle a portion of the inflammatory exudate was torn off, the fibrin having been intermingled with it. After this removal the auricular surface of each segment of the mitral valve was seen to

be fringed with grayish-white outgrowths. They were quite soft and varied in size from a bead to a cherry, and were irregular, warty, or roughened in appearance. A few outgrowths were also seen on the ventricular surface of the valve segments, and some were attached to the chorda tendineæ. (Fig. 1.[1]) No extension of the inflammatory process was seen on the endocardium of the auricular or ventricular

FIG. 2

Tricuspid valve in longitudinal section. × 18 diam.

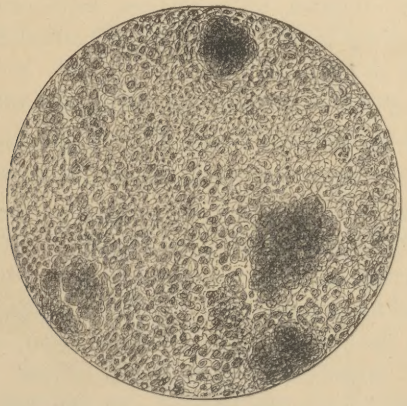
walls. On removal of the vegetations, a roughened appearance of the valve was observed, but no ulceration. The aortic valves were similarly encrusted with these vegetations, apparently springing from the corpora arantii. The pulmonary valves were normal. The aorta

[1] Three photographs and four micro-photographs, taken by Dr. Piersol, were exhibited. The photographs showed the outgrowths on the mitral, tricuspid, and aortic valves respectively. The micro-photographs showed sections of the valves and masses of micrococci, × 200 and × 700 respectively. The wood-cuts were cut from the photographs.

was healthy, save near its origin, where a few small spots of atheroma were detected; small aneurisms observed by others were absent.

Microscopical Examination.—The outgrowth examined was taken from the posterior curtain of the tricuspid valve. Its general appearances and arrangement, as seen in longitudinal section under low amplification, are accurately shown in the accompanying photo-micrograph. (Fig. 2.) The upper surface of the valve is covered with irregular, dense masses, forming a broken, but complete, investment. The under surface is generally free, some sections showing a few small areas of the same nature. By their intense and tenacious staining with methyl-blue, as well as by other characteristics, these masses

FIG. 3.



Masses of micrococci in the substance of the valve. × 200 diam.

appear composed of large zoöglœar tracts of micrococci. Examination under a power of 1000 diameters (Zeiss $\frac{1}{2\,0}$) confirms this supposition, suitable parts of the masses being readily resolved into the individual cocci. These masses of micrococci apparently rest *upon* the endothelium; in places, however, they penetrate to the subjacent tissue by processes, which recall strongly the penetrating pegs of epithelioma. The micrococci are arranged in groups, varying greatly in size, and generally distinctly limited. In several places, the micrococci occur in the connective tissue layer of the valve, as cir-

cumscribed, well-defined, oval masses, as seen in the photograph. (Fig. 3.) The subendothelial tissue in many fields is filled with diffused micrococci; the cell proliferation in this layer, however, being pronounced but in few places. The bulk of the growth is due to a very marked proliferation of the elements of the central, connective tissue layer of the valve. The fibres are separated and crowded apart by numerous small round and oval cells. On the under surface of the valve, about one and a half millimetre from the edge, exists a large oval mass—slightly less than one millimetre in diameter—composed principally of micrococci, among and dividing which are delicate septa, in connection with the delicate capsule surrounding the entire mass. Embracing this mass, and extending along the valve to and beyond its edge, is a thrombus. The surfaces of this are invested by a distinct membrane. Throughout the thrombus, among the well-preserved blood-cells, minute masses of micrococci are to be seen.

Examination of the wall of the left ventricle shows the muscular tissue itself to be unchanged; in the intermuscular connective tissue, especially near the endocardium, however, there is an increase in cellular elements.

Abdominal Cavity.—Liver weighs four pounds ten ounces. Fatty and slightly cirrhotic. Gall-bladder and ducts normal. *Spleen* weighs one pound five ounces, soft and pulpy. Malpighian bodies very marked, capsule thick and opaque. Neither organ contained hemorrhagic infarcts. *Kidneys*, each weigh ten ounces. Both have the appearance of the large white kidney. In the left a small cyst is seen, and in the right a very large hemorrhagic infarct.

Microscopical Examination of the Kidney.—The intertubular connective tissue is everywhere increased, and contains great numbers of small cells, this being especially marked around the larger blood-vessels and Malpighian bodies. These latter present conspicuous changes. From a condition where the glomeruli are simply swollen, distending the capsule, with, perhaps, an increase of nuclei, all stages of glomerulitis may be found—the capsule disappearing in the surrounding mass of inflammatory cells; finally, the complete atrophy of the Malpighian body. The uriniferous tubules of the labyrinth are distended with swollen granular cells; frequently they are occu-

pied with plugs of degenerated epithelium, blood-cells, and granular débris. The bloodvessels are engorged; in places near the Malpighian bodies partially absorbed extravasations are to be noticed. The larger vessels show some thickening of their walls. In a few vessels, in transverse section, micrococci were distinctly seen entangled among the blood-cells and proliferated endothelium.

Remarks.—During the period of observation—viz., ten weeks—the sequence of events was, acute articular rheumatism, the symptoms of which ameliorated in one week; a period of uncertain convalescence characterized by profuse night sweats; a recurrence of the rheumatism after ten days, with coincident endocarditis; a second remission of the joint symptoms, of twenty days duration, during which a pneumonia ran its course to almost complete resolution; a third relapse of rheumatic symptoms, with aggravation of the endocardial mischief, terminating fatally.

The somewhat meagre clinical history above detailed represents to a fair degree the course of infectious endocarditis. The points of especial interest are worthy of more distinct mention, and are as follows:—

a. The cause of the disease in this case was undoubtedly rheumatism. There was no focus of infection in any portion of the body. This is contrary to the general rule, for the cases analyzed by Prof. Osler more frequently originated from other causes than rheumatism.

b. The occurrence of pneumonia in the course of the disease is worthy of remark. Frequently pneumonia occurs synchronously with the development of the cardiac inflammation, or even anterior to that process; so that the question has been discussed whether the pneumonia is not the primary infective process. Developing

in the middle period of the disease, our case would lead one to infer that it was probably an accidental complication, more liable to occur on account of the disturbances in the circulation; markedly more so, if the right heart, as in our case, was involved.

c. The usual characteristic symptoms were present to a high degree,—intermitting fever, sweats, diarrhœa, dyspnœa, and rapid pulse. The course of the fever was so distinct and is so instructive that it is well worth while to give it in full. It is characterized by marked intermittency. It is appended with the record of the pulse and respiration the last three weeks of the patient's illness.

Record of temperature.

	A. M.	P. M.	
Oct. 3,		104°	
" 4,	102⅖°	102⅘°	
" 5,	102⅗°	102⅘°	
" 6,	102°	103⅗°	
" 7,	102°	98⅖°	
" 8,	99⅖°	100⅕°	
" 9,	97⅘°	97⅗°	
" 10,	96⅗°	96⅘°	
" 11,	96⅖°	99°	
" 12,	98°	98°	
" 13,	97°	99⅗°	
" 14,	98⅗°	99⅖°	
" 15,	99⅖°	101°	
" 16,	98⅖°	100⅕°	
" 17,	97⅘°	99⅖°	
" 18.	98⅖°	100⅗°	
" 19,	99°	101⅗°	
" 20,	101⅗°	100⅗°	
" 21,	102⅖°	100⅘°	Endocarditis.
" 22,	100⅕°	100⅗°	
" 23,	100°	100⅗°	
" 24,	100⅖°	102⅖°	
" 25,	101⅕°	100⅘°	
" 26,	100⅗°	101⅖°	

			A. M.			P. M.	
Oct. 27,			101⅖°			101⅗°	
" 28,			102⅗°			98⅗°	
" 29,			100⅕°			100⅕°	
" 30,			100⅘°			102⅕°	
" 31,			101⅗°			98⅕°	
Nov. 1,			99⅘°			102⅖°	Pneumonia.
" 2,			99⅕°			102°	
" 3,			99⅕°			100⅕°	
" 4,			102⅗°			102⅘°	
" 5,			102⅗°			103⅗°	
" 6,			103⅕°			103°	
" 7,			101⅖°			101⅘°	
" 8,			99⅕°			104°	
" 9,			103°			101⅗°	
" 10,			102°			104°	
" 11,			98⅗°			98⅖°	
" 12,			99⅖°			103⅕°	
" 13,			101⅖°			99⅖°	
" 14,			98⅘°			98⅖°	
" 15,			98⅖°			101⅖°	
" 16,			101⅕°			100⅕°	
" 17,			101⅘°			102⅕°	
" 18,			101⅘°			102⅕°	
" 19,			102°			103⅗°	
" 20,	Resp.	Pulse.	101⅘°	Resp.	Pulse.	98⅖°	
" 21,	28	124	102⅕°	32	140	103⅘°	
" 22,	25	100	96⅖°	30	112	99⅕°	
" 23,	28	124	102°	30	117	101⅗°	
" 24,	32	112	101⅗°	28	115	103⅖°	
" 25,	28	108	101⅕°	20	100	101⅗°	
" 26,	28	108	101⅕°	28	148	102⅗°	
" 27,	24	100	100⅘°	26	106	102⅘°	
" 28,	30	121	100⅘°	34	112	102⅗°	
" 29,	30	112	101⅘°	40	118	102°	
" 30,	28	100	100⅗°	38	156	103⅗°	
Dec. 1,	28	110	99⅘°	24	114	98⅗°	
" 2,	28	108	99⅕°	44	132	102⅕°	
" 3,	32	120	102°	34	140	103⅖°	
" 4,	30	120	101⅘°	30	126	103⅗°	
" 5,	21	106	100⅘°	32	144	103⅗°	
" 6,	30	130	103°	38	168	103⅗°	
" 7,	30	135	103⅖°	48	150	103⅗°	
" 8,	42	150	104°	44	138	103⅗°	
" 9,	36	120	103⅗°				

d. The appearance of the inflammatory lesions is remarkable. It was found on careful examination that no actual ulceration took place, but that the process was proliferative in nature, it being represented by the outgrowths on the valves.

Lancereaux described some cases which presented this appearance. Attention should be called to the occurrence of micrococci in the inflammatory products, not only of the endocardium, but in the glomeruli of the kidneys as well. The occurrence of the inflammation in the right ventricle is also worthy of remark. The absence of inflammation in this cavity has been said to be due to the absence of oxygenated blood. This case, as well as others of a like nature, certainly shows that a septic inflammation, to say the least, can take place in this locality.

Acute Pericarditis.—The clinical interest, otherwise attached to the next case, is somewhat detracted from by the short period it was under observation. It was, no doubt, originally a case of uræmia secondary to interstitial nephritis. The pericarditis developed in the course of the uræmia superinduced by exposure incident to removal to the hospital. One might consider the cerebral symptoms attributed to the uræmia, due to the pericarditis. The subnormal or normal temperature, the absence of physical signs of pericarditis on admission, and the absence of sufficient lesions in the pericardium to cause death (excessive fluid, etc.), leads one to infer that the uræmia preceded the local inflammation, and caused the death of our patient. In addition to the above points of interest, the age of the patient, and the absence apparently of sufficient cause for so grave organic disease of the kidneys are worthy of attention.

Case.—J. H., female, æt. 35, white, servant, admitted to hospital under care of Dr. Ludlow, December 23, 1884.

No knowledge of her previous history could be obtained, save that she had had some œdema of face and ankles, and an obstinate vomiting for a few days.

When admitted she was seen to be anæmic, but not emaciated, and that her face was puffy and her ankles swollen. The vomiting of a clear fluid continued without nausea. She did not complain of headache, but was dull, and slightly delirious. Heart normal; lungs congested posteriorly. Urine, spec. grav. 1004, highly albuminous with small granular casts; quantity in twenty-four hours, thirty-eight ounces.

Dec. 24.—Condition same. In the evening, a faint, double friction sound heard over the base of the heart.

25*th.*—Friction sound louder, and more distinct. Fremitus. No cardiac pain.

26*th.*—Roughened character to friction sounds. Vomiting continues. Temperature 98° P.M. No rise above since admission. Stupor.

27*th.*—Convulsions. Coma. Death.

Autopsy, nineteen hours after death.—Rigor mortis marked; œdema of legs and face. Pallid countenance. *Heart*, fourteen ounces, hypertrophied. Parietal and visceral layer of pericardium strewn with flakes of recent lymph, and on heart small villous outgrowths. No blood effusion or ecchymoses. No increase of serum. On opening heart, valves found normal, save small inflammatory patch on one leaflet of mitral valve. Atheroma of small amount first six inches of aorta. *Lungs* slightly œdematous. *Liver* congested and fatty; gall-bladder full of black bile; ducts patent. *Stomach* congested and mamillated. *Duodenum* congested and stained with bile. *Kidneys* weigh two and two and one-half ounces, respectively; capsules thickened and opaque; removed with difficulty. Cortical portion reduced in size and irregular; about junction of cortical and medullary portion, white striæ parallel to the medullary rays, looking not unlike deposits of calcareous matter, but which did not respond to the usual tests.

Microscopical Examination of the Kidney.—Very evident changes apparent throughout the organ. The capsule is thickened and adherent, its inner layers being infiltrated with small round cells. The connective tissue is increased, and its elements are actively engaged in proliferation, some fields being almost continuous tracts of closely crowded small round cells. The Malpighian bodies are generally in some stage of functional decline—from the beginning thickening of the capsule to final atrophy, many showing a complete amyloid degeneration. The cells of the uriniferous tubules are swollen, granular, or fatty. Many tubules contain waxy casts, and certain of the straight collecting tubules are impacted with granular fatty masses of desquamated cells—these tubules probably accounting for the whitish striæ noticed macroscopically. Many of the larger collecting tubules are entirely destitute of lining cells. The bloodvessels have greatly thickened walls, the increase taking place in their median and adventitious coats.

Microscopical Examination of Pericardium.—Sections of a patch from the parietal pericardium show an irregular mass composed chiefly of small round cells, embedded in a partially fibrillated matrix, the mass assuming the form, on its free surface, of irregular papillary processes. The individual cells are but poorly defined, and central irregular tracts are undergoing degeneration. The subendothelial tissue is infiltrated with small cells, proliferation of the elements of the tissue being pronounced. Micrococci were not distinguished in this mass.

[After the reading of the preceding paper:—]

Dr. Da Costa stated that he had listened with a great deal of interest to the report of the case of ulcerative endocarditis. It is certainly not a common affection in this country. Contrary to the usual opinion, in the instances which he had seen, there was mostly an association with acute rheumatism. The combination with pneumonia, on which some excellent observers have laid great stress, he had not often met with.

Dr. Musser said: I should like to make a remark in reference to the statistics in regard to rheumatism and pneumonia in ulcerative endocarditis. As I stated, these statistics are those of Dr. Osler, found in his article on infectious or ulcerative endocarditis. (*Transactions Internat. Med. Congress*, 1881.) I believe that he collected one hundred and fifty or sixty cases, in only one-third of which was rheumatism present. In the other cases, there was pneumonia or some other primary source of infection.

He has since told me that he has found this form of ulcerative endocarditis to be excessively frequent in pneumonia occurring as a complication. I am not able to give exactly his figures, but I think that in one hundred and three cases of pneumonia which he had examined, he had found it sixteen times.